

C Unit 6 C_{PRIVATE}

Risk Assessment and Communication

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6.1 INTRODUCTION

Risk assessments must be performed at hazardous waste or material sites in order to properly identify potential, long term risks to human health and the environment. Using epidemiological studies and toxicological information, dose/response relationships are calculated to estimate the risk or potential risk for all biota potentially exposed at the site. Formerly referred to as "endangerment assessments", and focused solely on human health risks, the EPA has incorporated assessments of risks to the environment into the risk assessment process.

6.1.1 RISK ASSESSMENT AND RISK MANAGEMENT

The National Research Council defines risk assessment as the use of a factual base to define the health effect of exposure of individuals or populations to hazardous materials or situations. It is distinguished from risk management, which is defined as the process of weighing policy alternatives and selecting the most appropriate regulatory action, integrating the results of risk assessment with engineering data and with social, economical, and political concerns to reach a decision regarding site remediation. The risk management decisions are made after the risk assessment has been completed.

The risk assessment process has been applied to the CERCLA ("Superfund") remedial investigation and feasibility study (RI/FS) format. The Superfund program established the methodology for characterizing the nature and estimating the extent of risks posed by uncontrolled hazardous waste sites and for developing and evaluating remedial options. The Superfund's National Contingency Plan is the implementation of the EPA mandate to assess the risks associated with hazardous material sites. The risk assessment process charges the site specific Remedial Project Manager (RPM) with identifying the impact of the hazardous materials. The risk assessment emphasizes protection of human health and the environment over the long term up to 70 years, and helps determine if action has to be taken to minimize untreated waste. This question needs to be asked in reference to concern over present exposure in addition to possible future exposures. This document utilizes CERCLA-format risk assessment procedures; similar procedures may be used by various other federal, state and industry organizations.

6.1.2 PERSONNEL

Key individuals in Superfund site risk assessment/risk management team are described below.

- ° **Risk Assessor**—the individual or team of individuals who actually organizes and analyzes site data, develops exposure and risk calculations, and prepares human health evaluation (i.e. risk assessment) reports. Risk assessors for Superfund sites are frequently contractors to EPA, other federal agencies, states, or potentially responsible parties.
- ° **Risk Assessment Reviewer**—the individual or team of individuals within an EPA region who provides technical oversight and quality assurance review of human health evaluation activities.
- ° **Remedial Project Manager (RPM)**—the individual who manages and oversees all RI/FS activities, including the human health evaluation, for a site. The RPM is responsible for ensuring adequate evaluation of human health risks and for determining the level of resources to be committed to the human health evaluation.
- ° **Risk Manager**—the individual or group of individuals who serves as primary decision-maker for a site, generally regional Superfund management in consultation with the RPM and members of the technical staff. The identity of the risk manager may differ from region to region and for sites of varying complexity.

The technical staff consists of statisticians, and sampling and monitoring personnel who collect the data to be used in risk assessment calculations.

6.1.3 BACKGROUND

There is no clear scientific consensus on how best to study the health effects attributable to environmental contamination. Formidable logistical and methodological obstacles attend all such efforts. Site-specific investigations require that information of many different kinds, usually gathered for purposes other than risk assessments (e.g., CERCLA RI/FS), be studied, evaluated, and integrated into a

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composite picture. Source termsC records or information about the identity and quantity of contaminants that originally entered the environmentC are often incomplete or unavailable. Exposure measuresC reliable ways of identifying who was exposed to the contaminants and in what amounts or patternsC are difficult to obtain. The populations involved are sometimes too small to demonstrate statistically significant differences in health status among exposed versus nonexposed groups. In some cases, "exposed" groups are diluted by individuals moving into and away from the site under study. Health outcomesC the biological effects to be investigatedC are also problematic. Current limitations on scientific understanding of toxicology make it difficult to associate specific symptoms, physical findings, or diseases with particular toxic exposures. Here again, small populations impose methodological barriers to reaching clear conclusions.

6.2 ECOLOGICAL ASSESSMENTS

Three types of information are needed to establish a firm, causal relationship between toxic wastes and ecological effects at hazardous waste sites (HWS). First, chemical analyses of the appropriate media are necessary to establish the presence, concentrations, and variabilities of specific toxic chemicals. Second, ecological surveys are necessary to establish that adverse ecological effects have occurred. And finally, toxicity tests are necessary to establish a link between the adverse ecological effects and the toxicity of the wastes. Without all three types of data, other potential causes of the observed effects unrelated to the toxic effects of hazardous wastes, such as habitat alterations and natural variability, cannot be eliminated. Confidence in cleanup and monitoring decisions is greatly enhanced when based on a combination of chemical, ecological, and toxicological data.

The objective of an ecological assessment is to estimate the ecological effects occurring at an HWS. Ecological effects refer principally to population- and community-level effects on terrestrial and aquatic biota and biological processes. The magnitude and extent of ecological effects are measured based on a select set of ecological endpoints that are considered reasonable indices of the status of biological populations and communities on and near HWSs.

The expected outputs from an ecological assessment include the following:

- ° a basic inventory of the current status of selected components of the biological community in the area;
- ° an estimate of the current level of ecological effects associated with the HWS based on the selected subset of

ecological endpoints;

- ° an estimate of the magnitude and variation of toxic effects; and
- ° to the degree possible, identification of the extent to which these effects have resulted specifically from the presence of hazardous and toxic chemicals, as opposed to other associated effects such as habitat disruption.

6.2.1 ASSESSMENT METHODS

The methods recommended for use in ecological assessments at HWSs are grouped into three major categories: (1) toxicity tests (2) biomarkers and (3) field surveys. Each of these basic methodologies contributes a different type of information to the HWS evaluation. As a result, all three must often be applied to gain a complete understanding of the ecological effects at an HWS. The following subsections provide an overview of the primary advantages, and also limitations, of each of these major categories of assessment methods.

6.2.1.1 Toxicity Tests

Toxicity tests measure the effects of contaminated media from the HWS on the survival, growth, and/or reproduction of aquatic and terrestrial biota. Most often, samples of soil, sediment, or water are collected from the HWS and returned to the laboratory for testing with several laboratory test species. Toxicity tests can also be run in mobile laboratories or in situ, and with resident species from the site.

The advantages and limitations of using toxicity tests in ecological assessments are reviewed in Table 1. Chemical analyses provide a measure of the total concentration of specific chemical compounds. Toxicity tests, on the other hand, provide an integrated index of the bioavailable toxic contaminants on the site. Furthermore, some toxic chemicals on a site may not be measured accurately in chemical analyses because of the complexity of the matrix or analytical detection limits. Thus, toxicity tests play an important role in and of themselves in site assessments, and potentially link the occurrence of contamination, as evidenced by an elevated chemical concentration, to biological effects. Toxicity tests are only an index, however, of the potential for population or community-level effects at the HWS. Demonstration and quantification of ecological effects require field

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surveys.

Results from toxicity tests are specific to the site of sample collection, and thus can be mapped to define gradients and zones of toxic conditions. Such maps, in addition to response surfaces of toxicity, can serve as a guide to the design of field surveys and other sampling programs.

Toxicity tests are generally classified as either acute (short-term) or chronic (long-term) depending on the length of exposure of the organism to the contaminated media. Acute toxicity tests are probably the best means for conducting a first-order assessment of the distribution and extent of toxic conditions at a site. They are relatively quick, easy, and inexpensive to conduct. On the other hand, acute tests tend to be less sensitive measures of toxicity than are chronic tests or biomarkers. Thus, the absence of an acute toxic response cannot be interpreted as the absence of a toxic problem. Chronic toxicity tests, while requiring additional time and expertise, may be needed to detect less severe, but still important, toxic effects. In particular, chronic toxicity tests may be used to define "no effect" levels, useful for evaluating the effectiveness of remediation programs.

6.2.1.2 Biomarkers

The term "biomarkers" refers to the measurement of selected endpoints in individual organisms, typically physiological or biochemical responses, that serve as sensitive indicators of exposure to contaminants and/or sublethal stress. As used in this document, measures of bioaccumulation, i.e., chemical concentrations of contaminants in organisms, are considered a biomarker of exposure.

TABLE 1

Advantages and Limitations of Toxicity Tests in Ecological Assessments

<u>Advantages</u>	
Measure of toxic conditions that can be linked to the presence of contaminants and hazardous wastes; an important assessment component needed to establish causality.	chemical analyses measure only total concentrations of specific compounds.
Results are an integrated index of bioavailable contamination, whereas	Results are specific to the location at which the sample was collected, thus they can be used to develop maps of the extent and distribution of bioavailable contamination and toxic conditions.

Limitations

Results are easily interpreted and amenable to QA/QC; within- and among-laboratory precision, estimates are already available for several tests.

Acute toxicity tests are relatively quick, easy, and inexpensive to conduct; results from acute tests are used as a guide in the design of chronic toxicity tests.

Chronic toxicity tests are generally more sensitive than are acute tests, and can be used to define "no effect" levels; in addition, chronic tests provide a better index of field population responses and more closely mimic actual exposures in the field.

Measure of potential toxic effects on resident biota at the HWS; however, cannot always be directly translated into an expected magnitude of effects on populations in the field.

Results are somewhat dependent on specific techniques, e.g., test species, water or soil quality, test duration, etc.

Ecological survey data also provide an integrated measure of effects for the entire HWS, and may be more useful for addressing certain assessments objectives.

Exposure conditions in toxicity tests are not directly comparable to field exposures; additional confounding variables and other stresses are important in the field.

Acute tests are less sensitive measures of toxic conditions (relative to chronic tests or biomarkers); thus, the absence of an acute toxic response cannot be interpreted as the absence of a toxicity problem

Chronic tests require more time and expertise to conduct, yet still may not detect all sublethal effects.

The advantages and limitations of using biomarkers in ecological assessments are reviewed in Table 2. An important advantage is their broad applicability. The techniques can be applied at many taxonomic levels (plants and animals) and the results have inferences that go beyond the organism(s) tested. Evidence for genotoxicity or disruption of basic physiological and biochemical

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processes based on biomarker analyses have relevance to assessments of potential hazards to human health.

Biomarkers can be measured in organisms collected from the field, reflecting "real-world" exposures, and in organisms exposed to contaminated media under more controlled conditions in the laboratory or in situ. Thus, biomarkers provide an important tool for comparing biological responses in the laboratory and in the field since the same methods can be applied in both environments. In addition, some tests are diagnostic of specific contaminants, and most tests provide some information on the mechanism of toxic response. All of these attributes aid in establishing causality for ecological effects in the HWS evaluation.

The major limitation in applying biomarkers in ecological assessments is the current lack of accepted, standardized, and tested markers for many of the HWS contaminants of interest. In addition, for most biomarkers, the relationship between a measured biomarker response and population-level effects has not been defined. Biomarkers are highly sensitive indices of exposure and sublethal response, but, within the context of an ecological assessment, their relevance is most evident when biomarked studies are conducted jointly with toxicity testing and field surveys.

6.2.1.3 Field Surveys

Field surveys involve the measurement of the structural and functional characteristics of populations and communities at the HWS's.

The advantages and limitations of using field surveys in ecological assessments are reviewed in Table 3. While toxicity tests may infer potential population and community-level effects, field surveys are the only means for demonstrating actual population and community-level effects at the HWS. Survey data identify the "problem" and the extent of the problem. Organisms are exposed in the "real world," and measured effects represent an integrated response to the temporal and spatial variations in exposure and contaminant concentrations in the field. With survey data alone, however, the causes for observed effects are difficult to determine. As noted in the preceding sections, causality is established best by a combination of approaches, including chemical sampling, toxicity testing, biomarkers, and field surveys.

TABLE 2

Advantages and Limitations of Biomarkers in Ecological Assessments

Advantages

Broadly applicable; a measure of biological response that crosses taxonomic lines, including inferences to potential human health effects.

Provides insight into the potential mechanisms of contaminant effects; in many cases, biomarkers are diagnostic of specific contaminants.

Can be applied in both the laboratory and field, providing an important linkage between laboratory toxicity tests and effects in the field.

For field samples, biomarkers provide an important index of bioavailability with "real-world" exposures.

When applied correctly (i.e., a biomarker appropriate for the contaminants at the site) may be a very sensitive index of bioavailability and biological response.

Limitations

Relationship between biomarkers and population-level effects in the field are not well defined.

Biomarkers are still lacking for most of the compounds of interest at HWSs.

Require particular care in sample handling as well as added time and expense.

For mobile species, difficult to define "exposure;" may require destructive sampling.

Important to carefully define reference conditions, a problem common to all field studies.

Results from field surveys and measures of ecological status are often highly variable, reflecting the high degree of variability (both spatial and temporal) in natural communities and, in some cases (e.g., fish communities in lakes), the problems inherent in sampling the biological community. As a result of this high background variability, fairly extensive sampling may be needed to measure the ecological characteristics of interest with a sufficient level of precision to detect "effects" related to the HWS. Careful attention to sampling design is required to ensure that the survey results satisfy the objectives (and data quality objectives) of the HWS evaluation. Procedures for quality assurance/quality control exist for field

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surveys, but they are not nearly as well established or clear-cut as are protocols for other components of the ecological assessment.

TABLE 3

Advantages and Limitations of Field Surveys in Ecological Assessments

<u>Advantages</u>	<u>Limitations</u>
Characterizes the basic ecology of the site, identifying important resident species and community types; based on results from the field survey, relevant species for use in toxicity testing and biomarker analyses can be identified.	Results from field surveys may be highly variable, requiring extensive sampling to measure ecological status with sufficient precision for detection of effects; as a result, the absence of a measurable effect cannot always be interpreted as no effect.
Potentially demonstrates definitive ecological effects in the field, delineating zones of effect and no apparent effect.	With survey data alone, causes for observed effects are difficult to determine.
Field responses integrate temporal and spatial variations in exposure and contaminant concentrations.	Results represent only a snapshot of the ecological status at the time of the survey.
Information on the status of terrestrial vegetation can be obtained from aerial photographs, eliminating the need to visit the HWS to survey terrestrial vegetation.	Procedures for QA/QC are not well established; difficult to measure precision and accuracy.
Key questions of interest for ecological assessments at HWS and recommended approaches for addressing these questions are summarized in Table 4.	

6.3 HUMAN HEALTH ASSESSMENTS

Linking human exposure to an environmental contaminant to a particular human health effect requires tracing a long and complicated trail from the original source of a pollutant to the particular symptom, disease, or other biological end point suffered **Insert Table 4** by an individual or population. The trail may be years or even decades old, and documentation of the original source term may not be ideal. The course of a contaminant's progress may literally be underground, where its route and direction cannot be visualized directly, or the pollutant may have been dispersed by winds long ago. Once a chemical or radionuclide is loose in the environment, it can interact with other substances, change chemical form, become diluted, transfer from

one medium to another, piggyback on other substances that transport it long distances, or accumulate in geophysical sinks or in plants and animals. Tracking such escape routes, mapping the present whereabouts of the contaminant, and designing measures to contain or eliminate the pollution are the purposes behind the remedial investigation/feasibility study (RI/FS) and facility investigation (RFI) processes of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and Resource Conservation and Recovery Act (RCRA), respectively. Virtually every aspect of CERCLA cleanup efforts thus far have involved efforts to identify, trace, and quantify environmental contamination.

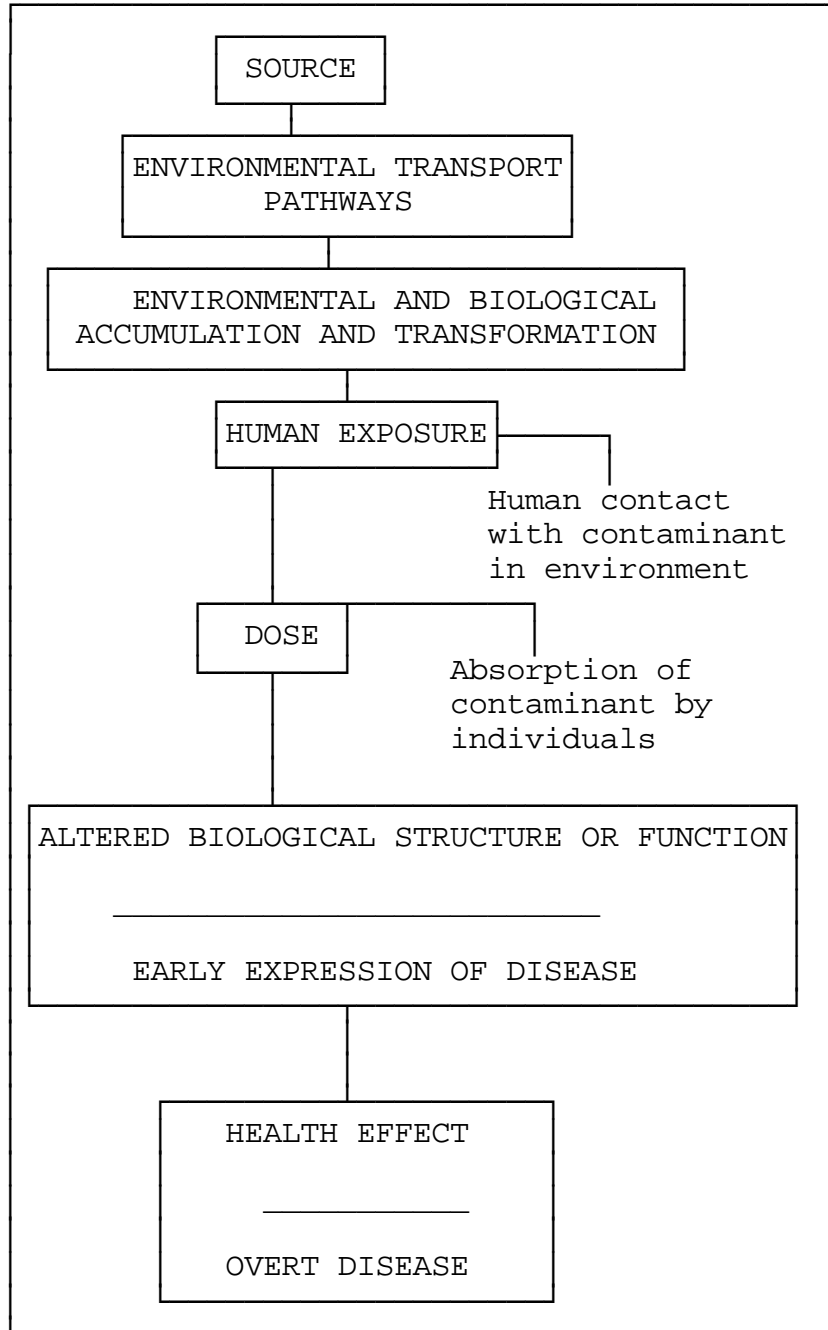
Figure 1 traces a contaminant from the source of pollution to the observable health effect. Most of the requirements stimulated by environmental regulations address the top half of the diagram: assessing the path and behavior of contaminants as they move into the environment and become potentially accessible to human contact. Very little effort has been directed toward investigating the *effects* of the contamination on human health or the environment.

Environmental health assessments focus on the bottom half of Figure 1, that part of the "toxic trail" leading from human exposure to health effect. For health investigators, information describing the types and whereabouts of the contamination is just the beginning of the puzzle. Environmental health assessments attempt to follow the progress of an environmental toxicant from its presence in the ambient environment at the point of potential human exposure through its absorption into the body and subsequent metabolism, accumulation, or elimination, and to relate these phenomena to observable expressions of dysfunction or to overt disease.

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FIGURE 1

Tracing the Toxic Trail



6.3.1 ANALYTICAL COMPONENTS OF ENVIRONMENTAL HEALTH ASSESSMENTS

Environmental health assessments must rely on what is known about the toxic effects of a chemical, or similar chemicals, to fill in the blanks and sketch tentative connections between exposure and disease. The aim of all analytical methods that seek to understand and predict the linkages between exposure to environmental agents and human health is to devise a legitimate means of relating a given exposure to a given biological effect *without* knowing all the terms in the bottom half of Figure 1.

All methods of assessing environmental health effects can be thought of as consisting of three key elements:

1. determining exposure and dose;
2. determining health effects; and
3. determining dose-response relationship (a term that quantitatively relates dose and effect).

The risk assessment process also includes analytical quantification of compounds present at the site, determination of reasonable/realistic exposure pathways, and duration of exposures.

Different environmental health assessment methodologies are distinguished by the ways in which these terms derived and utilized. Varying situations, purposes, and priorities may render some methods more suitable than others in identifying the terms and interpreting their meaning.

6.3.2 ENVIRONMENTAL EPIDEMIOLOGY

Epidemiology is the study of the occurrence and distribution of disease among populations. Epidemiology rests on the premise that disease does not occur randomly among populations but instead afflicts certain people at certain places and times according to the underlying causes of the illness. By studying these relationships, epidemiologists can achieve important insights into the association between certain exposures or risk factors and the occurrence of disease. Such insights can provide valuable tools in the prevention and control of disease.

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Environmental epidemiologic studies consist of several analytical components. For example, groups may be identified according to their exposure to a certain substance, operation, waste facility, etc. The occurrence of certain health outcomes, such as age-related mortality or cancer incidence among exposed groups, is investigated and compared with the health effects experienced by groups who were not exposed to the substance or process in question. If a positive association is discovered between a certain exposure and a particular health effect, and if the degree of exposure can be quantified, the results of epidemiological studies can be used to derive dose-response relationships, which in turn can be incorporated into quantitative risk analyses.

Epidemiological studies are less constrained by the limits of existing knowledge than are quantitative risk assessments (QRAs). In theory, the association between any exposure and any health effect could be examined by an epidemiological study; it is not necessary for such an exposure-effect linkage to have been previously noted or for dose-response data to be documented in the scientific literature. In contrast, quantitative risk assessments could not even consider the possible outcomes of complex exposures unless dose-response data were already available linking the exposure and the health effect in question.

For example, a quantitative risk assessment of the hazards of coke oven emissions would attempt to identify the health effects of exposure to each chemical ingredient, assess available dose-response information for each substance, quantify human exposure to each of these component chemicals, and sum the resulting chemical-specific cancer risk estimates. Not only would such a task require considerable effort, but the uncertainties, extrapolations, and data gaps would likely make it very difficult to reasonably or realistically project potential risks, with a modicum of scientific certainty (e.g., the genitourinary cancers observed in humans are not observed in animal experiments). One advantage of epidemiological studies is their ability to consider the health consequences of exposure to substances or combinations of substances whose toxic effects are now well understood.

The flexibility of epidemiology to focus on the particular toxic exposures and health effects of interest is offset by other methodological drawbacks, however. In conducting epidemiological studies, it is necessary to specify exposure accurately^C to identify who is exposed and who is not. If an appreciable number of people who are actually exposed are missed by investigators, or if exposed and nonexposed individuals are incorrectly labeled, the likelihood of detecting any true association between the exposure and the effect

decreases.

The difficulty of successfully documenting health outcomes among groups under study also plagues epidemiologists. If the occurrence of health effects over time cannot be tracked with accuracy because a significant portion of the exposed or nonexposed groups are "lost to follow-up," the chances of detecting any true association between the exposure and the effect decrease. These problems are often encountered when the health effects of interest occur only years or decades after initial exposure. Examples of such long-latency effects include cancers that appear 5 to 20 years after the exposures that caused them, and genetic defects that appear only in subsequent generations.

The detection of adverse health effects resulting from exposure to environmental toxicants is also complicated by individual variation in susceptibility to disease. Genetic factors, age, sex, the presence of underlying diseases, concomitant toxic exposures, and personal habits can all influence the expression of disease in an individual. Although such factors are difficult to identify and document, they can have a significant impact on the expression of overt illness among populations.

Quantitative estimation of the comparative measure of disease among exposed v. unexposed groups involves the use of statistical analyses. Statistical analyses endeavor to determine the likelihood that observed results are simply random events that do not truly indicate a real difference in baseline risk and, also, attempt to delineate (i.e., place "confidence intervals" around) the whole range of results compatible with the observed data. There are many opportunities for debate about which statistical strategies are appropriate for analyzing a given data set. Witness, for example, the long controversy over the risks associated with exposure to low-dose ionizing radiation.

The strength of the true association between the exposure and the health effect under study (i.e., the risk of disease if exposed compared with the risk without exposure) is the next important factor in determining whether an epidemiological study can detect "real" risks. If the risk of disease with exposure is very much greater than the risk of disease without it, large increases (i.e., exposure increases the baseline risk 10 to 100 times) may be detectable with

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small or moderate-size study populations. If, however, exposure increases the risk of disease by a factor of 10 or less, extremely large populations may have to be studied before adverse effects are detectable.

Herein lies another Achilles' heel of environmental epidemiology. Even if exposure can be documented adequately, epidemiology's lack of sensitivityCits inability to detect a small or moderate health effect even when the effect is truly presentClimits the usefulness of such studies. As one environmental health professional put it:

The definition of a public health disaster is an adverse effect so enormous that an epidemiological study can detect it.

6.3.3 EXPOSURE ASSESSMENT AND MISCLASSIFICATION

In infectious disease epidemiology, exposure is a relatively clear-cut concept; there are usually simple and reliable medical tests that identify the presence of antibodies, bacteria, or other markers of definite internal exposure or infection. Environmental epidemiologists, on the other hand, often have to make do with very crude measures of exposure. For example, "exposed" and "unexposed" groups might be determined by separating people into categories based on the distance between residence and a source of pollution. This is clearly not a very precise way of identifying true exposure status.

Incorrect assessments of who is and is not exposed to an environmental contaminant can have serious consequences for epidemiological investigations. Misclassification of exposure statusCincorrectly assigning people to "exposed" or "nonexposed" categoriesCcan obscure the actual association between exposure and the risk of adverse health effects. Misclassification always decreases the apparent risk of getting sick if one is exposed: the relative risk appears lower than it truly is. The effects of such misclassification become more serious as the true relative risk increases.

Biological markers are indicators of changes in cellular or biochemical components or processes, structure, or function that are measurable in biologic systems or samples. There are three types of biologic markers: those that indicate an organism's *exposure* to an exogenous substance, those that indicate an *effect* of such exposure, and markers that indicate *susceptibility* to an organism's ability to respond to an exposure. Biomarkers are desirable as indicators of exposure because, to the extent that they

are sensitive and specific, they permit assignment of individual exposure status and make misclassification errors less likely.

In practice, direct evidence of individual human exposure (i.e., evidence of actual contact between the pollutant and an individual) is rarely sought or measured when complying with environmental regulations or conducting quantitative risk assessments. Instead, indirect indicators, usually computer models of exposure estimates derived from environmental monitoring data, are used to estimate exposure. Advanced computer capabilities and analytic techniques are now available that permit reasonably accurate modeling of environmental transport pathways followed by various contaminants.

To complicate exposure assessments further, a long lag period or latency may occur between the time of exposure to an environmental toxicant and the manifestations of biological effects or the appearance of disease. Until the lag period has elapsed, health assessments of diseases, such as cancer, that are observed only after a latency of decades will not be informative. It is difficult, however, to accurately reconstruct exposures that occurred years or decades in the past.

Another issue that environmental health assessors must confront is the matter of exposure to combinations of contaminants. Communities located near nuclear weapons plants may be exposed to several environmental contaminants. The combined effects of such multiple exposures can be very different from the effects seen in response to individual contaminants. Once in the body, environmental toxins can accumulate, interact by impinging on the same organ system, or alter the metabolism of other toxins so that the biological impact of multiple exposure may differ significantly from the effect of exposure to individual substance. Investigators attempting to achieve a comprehensive picture of the health consequences of exposure to multiple contaminants must use professional judgment in anticipating what biological response(s) might result from such exposure burdens and exercise caution in selecting or rejecting which specific health effects to study.

Long-lived biomarkers could, in principle, provide an accurate reflection of integrated exposure patterns and cumulative exposure levels. Levels of polychlorinated biphenyls (PCBs), for instance,

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persist in human fat cells for decades. Samples of adipose tissue can, therefore, provide an estimate of long-term exposure to PCBs. In practice, however, few persistent biomarkers of exposure to environmental contaminants are available.

Another issue of relevance to the investigation of toxic exposures and their effects on populations is what statisticians call "variability." Exposures are seldom homogeneous through time and across populations. Instead, there are often episodic exposure excursions that may be many times greater than the average. Because the pattern and intensity of exposure are so important in determining biological effects, computations based on average monitoring data may fail to represent real-world conditions accurately. It is difficult to design laboratory experiments or computer models that mimic such episodic exposure patterns.

6.3.4 QUANTITATIVE RISK ASSESSMENTS

Quantitative risk assessments typically consist of at least four steps:

1. hazard identification;
2. dose-response assessment;
3. exposure assessment; and
4. risk characterization.

Hazard identification is the determination of whether a substance causes adverse biological effects. The Environmental Protection Agency (EPA) uses a "weight of evidence" approach in judging the hazard potential of a substance. All available scientific evidence is reviewed and evaluated for accuracy, applicability, etc., so that the most suitable data are used to assess the nature of the hazard posed by a chemical. The effects of substances that are structurally similar may be considered. Most available toxicological information comes from animal experiments. In the absence of a compelling reason to evaluate the hazards of a particular mixture, only individual contaminants are considered. If a substance is determined to be nonhazardous, or no data are available indicating that the substance is hazardous, the risk assessment ends here.

At Superfund sites, "indicator chemicals" are selected from lists of contaminants revealed by preliminary analysis to be present at the site. Indicator chemicals are those believed to pose the greatest health hazard at a site; they are chosen on the basis of toxicity (i.e., hazard identification), concentration and amount, mobility, frequency of detection, and persistence in the environment. At more complex sites a proportionally larger number of indicator chemicals should be examined.

Dose-response assessments specify the quantitative relationship between a given dose (absorbed amount) of a substance and the severity or probability of an adverse effect; they provide a measure of a substance's potency. Selection of a dose-response relationship can be controversial, in part because the interpretation of most available data requires extrapolation across several categories, usually including species, sex, age, dose range, exposure pattern, and absorption routes. Human data derived from epidemiological studies are allotted more weight when available, but most epidemiological studies focus on occupational exposures and situations that are not necessarily representative of environmental exposures in the general population. Deriving dose-response relationships of low dosages of potentially carcinogenic substances may be especially controversial because available data can often be reconciled with more than one mathematical dose-response model.

Exposure assessments are estimates of the degree of individual exposure to a given substance and the number of people exposed. The determination of exposure is crucial in conducting quantitative risk assessments. If the actual or potential exposure is not recognized, either because of failure to identify significant environmental transport pathways and exposure routes or because of inaccurate estimation of the number of people exposed or exposure levels, the resulting risk estimate will be useless.

Direct measurements of human exposure (e.g., analyses of blood or urine samples that indicate individual exposure to a substance) are rarely used in QRAs, and most such measures remain research tools. Instead, QRAs typically use indirect measures of human exposure to project estimates of individual dose. For example, some measure (mean, median, or upper confidence limit levels) of ambient contaminant concentrations may be multiplied by standard intake values (estimates of how much air one breathes, water one drinks, etc., over a 70-year lifetime or other appropriate exposure duration) to produce a dose estimate. The estimated dose is then related to the relevant legal standards or to exposure levels that have been predicted to pose no more than "acceptable" levels of risk for the health effect at issue.

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6.4 RISK ASSESSMENT PROCEDURES

Risk assessment procedures for either ecological or human health assessments follow essentially the same format. Differences between the two are present due to substantial differences in the body of knowledge about toxicity levels for chemicals in humans versus animals. Ecological knowledge deals with systems; relatively little is known about the precise effect of a given chemical on a particular species.

This section explains the basic procedure for conducting assessments for human health risks. Variations for ecological risk assessments may be inferred. The flow chart below illustrates the essential steps in risk assessments. Each step is discussed in detail.

6.4.1 DATA COLLECTION

In order to properly gauge risk to an environment, assessors must first have a body of knowledge about the environment that they are assessing. Data about the site must be collected, including air, water, and soil samples in order to determine the scope and range on any on-site contamination. Preliminary RI/FS scoping should establish modeling parameter needs prior to any on-site data collection.

Table 5 provides examples of the modeling parameters for which information may need to be obtained during a site sampling investigation. Note that separate and distinct parameters are identified for each media in addition to general source characteristic parameters.

Quantitative risk assessment requires data on concentrations of contaminants in each of the source areas and medias of concern. Background sampling is conducted to distinguish site-related contamination from naturally-occurring or other non-site-related levels of chemicals. There are two different types of background levels of chemicals:

- ° naturally occurring levels, which are ambient concentrations of chemicals present in the environment that have not been influenced by humans; and
- ° anthropogenic levels, which are concentrations of chemicals that are present in the environment due to human-made, non-site sources (e.g., industry, automobiles).

6.4.1.1 Preliminary Exposure Assessment

A preliminary identification of potential human exposure provides much needed information for the Sampling and Analysis Plan. This activity involves the identification of:

TABLE 5

Examples of Modeling Parameters

Type of Modeling	Modeling Parameters ^a
Source Characteristics	Geometry, physical/chemical Conditions, emission rate, emission strength, geography
Soil	Particle size, dry weight, pH, redox potential, mineral class, organic carbon and clay content, bulk density, soil porosity
Ground-water	Head measurements, hydraulic conductivity (pump and slug test results), saturated thickness of aquifer, hydraulic gradient, pH, redox potential, soil-water partitioning
Air	Prevailing wind direction, wind speeds, stability class, topography, depth of waste, contaminant concentration in soil and soil gas, fraction organic content of soils, silt content of soils, percent vegetation, bulk density of soil, soil porosity
Surface Water	Hardness, pH, redox potential, dissolved oxygen, salinity, temperature, conductivity, total suspended solids, flow rates and depths for rivers/streams, estuary and embayment parameters such as tidal cycle, saltwater incursion extent depth and area, lake parameters such as area, volume, depth, depth to thermocline
Sediment	Particle size distribution, organic content, pH, benthic oxygen conditions, water content
Biota	Dry weight, whole body, specific organ, and/or edible portion chemical concentrations, percent moisture, lipid content, size/age, life history stage

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^a These parameters are not necessarily limited to the type of modeling with which they are associated in this exhibit. For example, many of the parameters listed for surface water are also appropriate for sediments.

- ° media of concern
 - C currently contaminated media
 - C currently uncontaminated media
- ° areas of concern
 - C locations of samples to be collected
- ° types of chemicals expected at the site
- ° potential routes of contaminant transport through the environment

Identification of these potentials will help in creating a sampling plan that will most accurately assess the human health and environmental risks that the site presents.

6.4.1.2 Sampling Strategy

The overall strategy for sample collection must be devised, precisely defining sample size, type, and location. Typically, sample size and sample location are determined at the same time. A number of considerations are associated with determining an appropriate number of samples for a risk assessment. These considerations include:

- ° number of areas of concern that will be sampled
- ° statistical methods that are planned
- ° statistical performance (i.e., variability power, and certainty) of the data that will be collected, and
- ° practical considerations of logistics and cost

There are three general strategies for establishing sample locations:

- ° **Purposive.** Sampling locations within the areas of concern generally should not be sampled purposively if the data are to be used to provide defensible information for a risk assessment.
- ° **Random.** Random sampling involves selecting sampling locations in an unbiased manner.
- ° **Systematic.** Systematic sampling locations are established across an area of concern by laying out a grid of sampling locations that follow a regular pattern.

The types of samples to be collected must also be determined. Two types are commonly used: grab and composite. Grab samples

represent a single unique part of a medium collected at a specific locations and time. Composite samples combine subsamples from different locations and/or times.

6.4.1.3 Quality Assurance/Quality Control (QA/QC)

QA/QC considerations that are of particular importance for risk assessment sampling include sampling protocol, sampling devices, QC samples, collection procedures, and sample preservation. The risk assessor is not responsible for QA/QC evaluations, but should be aware of these considerations and include them in the sampling plan.

6.4.1.4 Risk Assessor Role

The risk assessor should be sure to take an active role during workplan development and data collection. This role involves three main steps:

- ° present risk assessment sampling needs at RI/FS scoping meeting;
- ° contribute to the workplan and review the Sampling and Analysis Plan; and
- ° conduct interim reviews of outputs of the field investigation.

6.4.2 DATA EVALUATION

After a site sampling investigation has been completed, a large quantity of analytical data is usually available. Each sample may have been analyzed for over one hundred chemicals, and many of those chemicals may have been detected. The following steps should be followed to organize the data into a form appropriate for a baseline assessment:

- ° combine data available from site assessments and sort by medium;
- ° evaluate analytical methods;
- ° evaluate the quality of the data with respect to sample quantitation limits;
- ° evaluate quality of the data with respect to qualifiers and codes;

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- evaluate the quality of the data with respect to the blanks;
- evaluate tentatively identified compounds;
- compare potential site-related contamination with background;
- identify chemicals of potential concern;
- develop a set of data for use in the risk assessment; and
- if appropriate, further limit the number of chemicals to be carried through the risk assessment.

A flow chart of this evaluation process is presented in Figure 6.3. The outcome of this evaluation is (1) the identification of a set of chemicals that are likely to be site-related and (2) reported concentrations that are of acceptable quality for use in the quantitative risk assessment. If the data evaluation steps are followed, the number of chemicals to be considered in the remainder of the risk assessment usually will be less than the number of chemicals initially identified. Chemicals remaining in the quantitative risk assessment based upon this evaluation are referred to as "chemicals of potential concern".

In selecting the data to include in the risk assessment, the objective of the risk assessor is to characterize as accurately as possible the extent of contamination.

Data summary tables should be developed for each medium sampled. Summary statistics are crucial to the selection of contaminants of concern for the risk assessment. Each data summary table should indicate the frequency of detection, observed range of concentration, and the mean and maximum value for each contaminant detected in each media. Most importantly, the format and the method used to summarize the information should be clear and consistent across all environmental media.

Either the arithmetic or geometric mean may be used as a statistical method to summarize data. Usually, an arithmetic mean is used for normally-distributed data, while a geometric mean is preferred for log-normally distributed data to minimize the influence of outlying data points. Regardless of the statistical method selected, the mean calculation should be consistent across all environmental media. Additionally, the rationale behind the selection of a particular summary statistical method should be discussed in the Hazard Identification.

6.4.3 EXPOSURE ASSESSMENT

The objective of the exposure assessment is to estimate the type and magnitude of exposures to the chemicals of potential concern that are present at or migrating from a site. The results of the exposure assessment are combined with chemical-specific toxicity information to characterize potential risks.

The magnitude of exposure is determined by measuring or estimating the amount of an agent available at the exchange boundaries (i.e., the lungs, gut, skin) during a specific time period. Exposure assessment is the determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, or route of exposure. Exposure assessments may consider past, present and future exposures, using various techniques for each phase. Estimates of current exposures can be based on measurements or models of existing conditions, and those of past exposures can be based on measured or modeled past concentrations or measured chemical concentrations in tissues. If human monitoring is planned to assess current or past exposures, the Agency for Toxic Substances and Disease Registry (ATSDR) should be consulted to take the lead in conducting these studies and assessing the current health status of the people near the site based on the monitoring results.

The exposure assessment proceeds with the following steps:

- ° **Characterization of Exposure Setting.** The general physical characteristics of the site and the characteristics of the populations on or near the site
- ° **Identification of Exposure Pathways.** Those pathways by which the previously identified populations may be exposed.
- ° **Quantification of Exposure.** The magnitude, frequency and duration of exposure for each pathway is estimated. This step is most often conducted in two stages:
 - C estimation of exposure concentrations
 - C calculation of intakes

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In general, a great deal of professional judgement is required to estimate exposure concentrations. Exposure concentrations may be estimated by (1) using monitoring data alone, or (2) using a combination of monitoring data and environmental fate and transport models. In most exposure assessments, some combinations of monitoring data and environmental modeling will be required to estimate exposure concentrations.

6.4.3.1 Monitoring Data

Use of monitoring data to estimate exposure concentrations is normally applicable where exposure involves direct contact with the monitored medium, or in some cases where monitoring has occurred directly at the exposure point (e.g., a residential drinking water well). For these exposure pathways, monitoring data generally provide the best estimate of current exposure concentrations.

6.4.3.2 Fate and Transport Models

In some instances it may not be appropriate to use monitoring data alone, and fate and transport models may be required to estimate exposure concentrations. Specific instances where monitoring data alone may not be adequate are as follows:

- ° where exposure points are spatially separate from monitoring points;
- ° where temporal distribution of data is lacking; and
- ° where monitoring data are restricted by the limit of concentration.

A wide variety of models are available for use in exposure assessments.

The level of effort to be expended in estimating exposure concentrations will depend on the type and quantity of data available, the level of detail required in the assessment, and the resources available for the assessment. In general, estimating exposure concentrations will involve analysis of site monitoring data and application of simple, screening-level analytical models. The most important factor in determining the level of effort will be the quantity and quality of the available data. In general, larger data sets will support the use of more sophisticated models.

6.4.3.3 Calculation of Intakes

Following the estimation of exposure concentrations, the chemical-specific intakes for the populations and exposure pathways selected for quantitative analysis must be calculated. The general equation for estimating intake is shown in Figure 2. Remember that intakes calculated in this step are expressed as the amount of chemical at the exchange boundary and available for absorption. Intake, therefore, is not equivalent to absorbed dose, which is the amount of chemical absorbed into the blood stream.

Figure 3 is an example of the standard equation for estimating human intakes for residential exposure from the ingestion of chemicals found in drinking water. Other formulas exist for other routes and pathways of exposure such as occupational exposures, and dermal contact with soil, air and water. It is important to remember that formulas for each possible route of exposure for each chemical of concern must be calculated in order to accurately represent the health risk.

6.4.4 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for particular contaminants to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a contaminant and the increased likelihood and/or severity of adverse effects.

Toxicity assessment is an integral part of the overall risk assessment. although toxicity information is critical to the risk assessment, the amount of new research required to complete this step is limited in most cases. EPA has performed the toxicity assessment step for many chemicals and has made available the resulting toxicity information and toxicity values, which have undergone extensive peer review. At some sites, however, there will be significant data analysis and interpretation issues that should be addressed by an experienced toxicologist.

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FIGURE 2
Generic Equation for Calculating Chemical Intakes

$$I = C \times \frac{CR \times EFD}{BW} \times \frac{1}{AT}$$

Where:

I = intake; the amount of chemical at the exchange boundary (mg/kg body weight-day)

Chemical-related variable

C = chemical concentration; the average concentration contacted over the exposure period (e.g., mg/liter water)

Variables that describe the exposed population

CR = contact rate; the amount of contaminated medium contacted per unit or event (e.g., liters/day)

EFD = Exposure frequency and duration; describes how long and how often exposure occurs. Often calculated using two terms (EF and ED):

EF = exposure frequency (days/year)

ED = exposure duration (years)

BW = body weight; the average body weight over the exposure period (kg)

Assessment-determined variable

AT = averaging time; period over which exposure is averaged (days)

FIGURE 3

Residential Exposure: Ingestion of Chemicals in Drinking Water (and beverages made using drinking water)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CW} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

CW = Chemical Concentration in Water
(mg/liter)

IR = Ingestion Rate (liters/day)

EF = Exposure Frequency (days/year)

ED = Exposure Duration (years)

BW = Body Weight (kg)

At = Averaging Time (period over which exposure is averaged --days)

Variable Values:

CW: Site-specific measured or modeled value

IR: 2 liters/day (adult, 90th percentile; EPA 1989d)
1.4 liters/day (adult, average; EPA 1989d)
Age-specific values (EPA 1989d)

EF: Pathway-specific value (for residents, usually daily --365 days/year)

ED: 70 years (lifetime; by convention)
30 years (national upper-bound time (90th percentile) at one residence; EPA 1989d)
9 years (national median time (50th percentile) at one residence; EPA 1989d)

BW: 70 kg (adult, average; EPA 1989d)
Age-specific values (EPA 1985a, 1989d)

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AT: Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 year x 365 days/year).

Toxicity assessments often use bioassay to gauge the degree of toxicity in humans and other biota. Bioassay is a method for quantitatively determining the concentration of a substance by its effect on the growth of a suitable animal, plant, or microorganism under controlled conditions. Usually biota known to be sensitive to particular chemicals are used in these controlled conditions to assess a chemical of unknown toxicity of the same family. Tests may be conducted in vitro, in an artificial apparatus, or in vivo, in the living cell or organism.

6.4.4.1 Hazard Identification

Hazard identification is the process of determining whether exposure to an agent can cause an increase in the incidence of a particular adverse health effect (e.g., cancer, birth defect) and whether the adverse health effect is likely to occur in humans. Hazard identification involves characterizing the nature and strength of the evidence of causation.

6.4.4.2 Dose-Response Evaluation

The second step of the toxicity assessment, dose-response evaluation, is the process of quantitatively evaluating toxicity information and characterizing the relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity values (e.g., reference doses, RfD, and slope factors) are derived that can be used to estimate the incidence or potential for adverse effects as function of human exposure to the agent. These toxicity values are used in the risk characterization step to estimate the likelihood of adverse effects occurring in humans at different exposure levels.

6.4.4.3 Steps in Toxicity Assessment

The initial step to be taken in a toxicity assessment includes gathering toxicity information, qualitative and quantitative, for the substances being evaluated. Because EPA has researched the toxicity of many chemicals already, it is wise to utilize these sources

of toxicity information first.

In the first step of the toxicity assessment, information is collected regarding the toxic effects that occur following exposure to the chemical being evaluated. Particular attention should be paid to the route of exposure, the frequency and length of exposure, and the doses at which the adverse effects are expected to occur. Chemicals having potential reproductive or developmental effects should be flagged. Later in the evaluation, special reference doses for developmental effects can be sought for these chemicals. Figure 7 summarizes the necessary steps to be taken in a toxicity assessment.

Several sources may provide useful toxicity information and references to primary literature, although only some of them should be used as sources for slope factors and reference doses (as explained below).

Integrated Risk Information System (IRIS). IRIS is an EPA data base containing up-to-date health risk and EPA regulatory information for numerous chemicals. IRIS contains only those RfDs and slope factors that have been verified by the RfD or CRAVE Workgroups and consequently, is considered to be the preferred source of toxicity information. Information in IRIS supersedes all other sources. Only if information is not available in IRIS for the chemical being evaluated should the sources below be consulted. IRIS consists of a collection of computer files on individual chemicals. Existing information on the chemicals is updated as new scientific data are reviewed. New files and new chemicals are added as information becomes available. These chemical files contain descriptive and quantitative information in the following categories:

- ° oral and inhalation chronic reference doses;
- ° oral and inhalation slope factors and unit risks for chronic exposure to carcinogens;
- ° Health Advisories from EPA's Office of Drinking Water;
- ° EPA regulatory action summaries; and
- ° supplemental data on acute health hazards and physical/ chemical properties.

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To ensure access to the most up-to-date chemical information, IRIS is only available on line. For information on how to access this data base, call IRIS User Support at 513-569-7254 or see the *Federal Register* notice regarding the availability of IRIS (EPA 1988a).

Should EPA regional staff have specific technical or scientific questions about any verification workgroup's analysis of particular data cited in IRIS, the Agency contact for a particular chemical (identified at the end of each IRIS file) should be consulted. If new data are identified suggesting that existing IRIS information may be outdated, or if there is concern or disagreement about the overall findings of particular files, the Agency IRIS coordinator should be consulted. The IRIS coordinator can assist in making arrangements should discussions with a verification workgroup be needed.

Health Effects Assessment Summary Tables (HEAST). Formerly "The Quarterly" and associated references, HEAST is a tabular presentation of toxicity information and values for chemicals for which Health Effects Assessments (HEAs), Health and Environmental Effects Documents (HEEDs), Health and Environmental Effects Profiles (HEEPs), Health Assessment Documents (HADs), or Ambient Air Quality Criteria Documents (AAQCDs) have been prepared. HEAST summarizes interim (and some verified) RfDs and slope factors as well as other toxicity information for specific chemicals. In addition, HEAST directs readers to the most current sources of supporting toxicity information through an extensive reference section. Therefore, HEAST is especially helpful when verified information for a chemical is not in IRIS. HEAST, which is updated quarterly, also provides a valuable pointer system for identifying current references on chemicals that are not in IRIS.

HEAST can be obtained upon request from the Superfund Docket (FTS or 202-382-3046). The Docket will mail copies of HEAST to callers and place requestors on a mailing list to receive an updated version quarterly. HEAs, HEEDs, HEEP, HADs, and AAQCDs referenced in HEAST are available through EPA's Center for Environmental Research Information (CERI) in Cincinnati, OH (513-569-7562 or FTS 684-7562) or the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161 (703-

487-4650 or 800-336-4700).

EPA criteria documents. These documents include drinking water criteria documents, drinking water Health Advisory summaries, ambient water quality criteria documents, and air quality criteria documents, and contain general toxicity information that can be used if information for a chemical is not available through IRIS or the HEAST references. Criteria documents are available through NTIS at the address given above. Information on drinking water criteria documents can be obtained through the Safe Drinking Water Hotline (800-426-4791).

Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profiles. ATSDR is developing toxicological profiles for 275 hazardous substances found at Superfund sites. The first 250 substances to be addressed have been identified in *Federal Register* notices (EPA 1987, 1988b, 1989, 1990). These profiles contain general toxicity information and levels of exposure associated with lethality, cancer, genotoxicity, neurotoxicity, developmental and reproductive toxicity, immunotoxicity, and systemic toxicity (i.e., hepatic, renal, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, and dermal/ocular effects). Health effects in humans and animals are discussed by exposure route (i.e., oral, inhalation, and dermal) and duration (i.e., acute, intermediate, and chronic). Also included in the profiles are chapters on physicochemical properties, environmental fate, potentials for human exposure, analytical methods, and regulatory and advisory status. Contact ATSDR for further information on the status or availability of a particular profile.

ATSDR
Division of Toxicology
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1600 Clifton Rd. NE
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Phone: (404) 639-6000
Fax: (404) 639-6060

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EPA's Environmental Criteria and Assessment Office (ECAO). ECAO may be contacted at 513-569-7300 (FTS 684-7300) for general toxicological information as well as for technical guidance concerning route-to-route extrapolations, toxicity values for dermal exposures, and the evaluation of chemicals without toxicity values. The requestor should identify their need for a "rapid response request" (within 48 hours) for interim guidance on Superfund health-related issues. Contractors must give the name and address of their RPM or regional risk assessment contact before ECAO will respond. RPMs and regional contacts will be sent a copy of ECAO's response to the contractor.

Open literature. A primary literature search may be valuable for determining whether new data are available that may affect IRIS information.

Because toxicity information may change rapidly and quickly become outdated, care should be taken to find the most recent information available. IRIS is updated monthly, provides verified RfDs and slope factors, and is the Agency's preferred source of toxicity information. Only if values are unavailable in IRIS should other information sources be consulted

HEAST is the second most current source of toxicity information of importance to Superfund. Unlike IRIS, HEAST provides information regarding interim as well as verified RfDs and slope factors. Readers are directed to supporting toxicity information for interim and verified values in an extensive reference section of HEAST. HEAST information should only be sought for those chemicals not listed in IRIS.

Toxicity information, RfDs, and slope factors also can be found in other EPS documents. Although these values were developed by offices within the Agency, they have not necessarily been verified by the RfD or CRAVE Workgroups. The use of up-to-date verified information is preferred to the use of interim information and, therefore, toxicity information should be obtained from other EPA references only if information could not be found in IRIS or HEAST. Before using references other than those cited in IRIS or HEAST, check with ECAO at 513-569-7300 (FTS 684-7300) to see if more current information is available.

6.4.4.4 Toxicity Assessment for Non-Carcinogenic Effects

A reference dose, or RfD, is the toxicity value used most often in evaluating noncarcinogenic effects resulting from contaminant exposures. Various types of RfDs are available depending on the exposure route (oral or inhalation), the critical effect (developmental or other), and the length of the exposure being evaluated (chronic, subchronic, or single event). EPA-verified RfDs are only for chronic exposures, and the EPA now refers to inhalation values as reference concentration (RFC).

The reference dose is derived from the following equation:

$$\text{RfD (mg/kg/day)} = \frac{\text{NOAEL or LOAEL}}{\text{U.F.}}$$

where the NOAEL (No Observable Adverse Effect Level) represents the dose of a chemical at which there is no statistically or biologically significant difference in frequency of an adverse effect between the exposed population and its appropriate control.

The LOAEL (Lowest Observable Adverse Effect Level) represents the lowest dose where a statistically significant difference in the frequency of an adverse effect is observed. The uncertainty factor (U.F.) is included to account for interspecies and intraspecies differences, severity of the adverse effect, and the adequacy of the data.

A chronic RfD is defined as an estimate (with uncertainty factors up to 1000) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposures to compound. Chronic RfDs generally should be used to evaluate the potential for noncarcinogenic effects associated with exposure periods between seven years and a lifetime.

Subchronic RfDs are useful for characterizing potential noncarcinogenic effects associated with shorter-term exposures, and developmental RfDs are useful for assessing potential developmental effects resulting from exposure to a compound.

Reference values that may be useful for evaluating potential

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adverse effects associated with oral exposures of shorter duration have been developed by the Office of Drinking Water. Like the MCLG, the one-day, ten-day, and lifetime Health Advisories are guidelines derived strictly on health considerations. They are based on a 10-kg child assumed to drink 1 liter of water per day, and a margin of safety is included to protect sensitive members of the population.

6.4.4.5 Toxicity Assessment for Carcinogenic Effects

A slope factor and the accompanying weight-of-evidence determination are the toxicity data most commonly used to evaluate potential human carcinogenic risks.

In the first step of the evaluation, the available data are evaluated to determine the likelihood that the agent is a human carcinogen. The evidence is characterized separately for human studies and animal studies as sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined, and based on the extent to which the agent has been shown to be a carcinogen in experimental animals or humans, or both, and any other supporting evidence of carcinogenicity, the agent is given a weight of evidence classification.

In the second part of the evaluation, based on the evaluation that the chemical is a known or probable human carcinogen, a toxicity value that defines quantitatively the relationship between dose and response (i.e., the slope factor) is calculated.

Generally, the slope factor is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. Slope factors should always be accompanied by the weight-of-evidence classification to indicate the strength of the evidence that the agent is a human carcinogen.

6.5 STEPS IN RISK CHARACTERIZATION

This section provides a brief overview of the risk characterization process. Risk characterization is the process wherein all the foregoing pieces are incorporated into a mathematical model that represents the probable risks of exposure to a given population for which a risk estimate is being calculated. Risk characterization is clearly dependent on the accuracy of its components: the applied

hazard information, dose-response relationships, and exposure estimates.

Risk characterization also requires judgments about how to handle uncertainties in the underlying data, how to select appropriate dose-response and exposure estimates from (often incomplete, ambiguous, or conflicting) available data, how to assemble this information into an overall model, and how to present the results of the assessment and its attendant uncertainty to the risk manager.

A risk assessment also establishes criteria for the Remedy Selection Process as part of the risk management decision. The risk assessor needs to judge how much exposure the individuals will incur, and how clean the environment will need to be to minimize the risk. The feasibility of attaining such levels must also be considered.

The purpose of the feasibility study (FS) is to provide the decision-makers with an assessment of remedial alternatives, including their relative strengths and weaknesses, and the trade-offs in selecting one alternative over another. The FS process involves developing a reasonable range of alternatives and analyzing these alternatives in detail.

The first step in the FS process involves developing remedial action objectives that address contaminants and media of concern, potential exposure pathways, and preliminary remediation goals. These goals are based initially on readily-available chemical-specific ARARs (e.g., MCLs for drinking water). Preliminary remediation goals for individual substances are refined or confirmed at the conclusion of the baseline risk assessment.

ARARs are site-specific requirements for remediation that take into consideration what is technically feasible and appropriate for cleanup activities. Risk assessments will be made independent of ARARs, but the selection of the remediation method will take both ARARs and the risk assessment analysis into consideration.

ARARs are "Applicable or Relevant and Appropriate Requirements", meaning:

applicableCrequirements specifying and addressing

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hazardous substances, pollutants, contaminants, remedial acts relevantCaddresses problems pertinent at conditions of site appropriateCrequirements well suited to particular site

MCLs are maximum contaminant levels for certain toxic substances found in drinking water. Established by the Safe Drinking Water Act, primary standards set limits on contaminants that may affect health such as fluoride, arsenic, a variety of pesticides, mercury, lead, nitrates, several additional organic and inorganic chemicals, and radionuclides.

Secondary levels are also established by the Act, specifying the MCLs consistent with protection of public welfare. These are advisory only, and include limits on various physical characteristics which may not harm health by may make water less pleasing to drink or use. Included in these limits are standards for chloride, copper, iron, and manganese.

Knowing these chemicals, and the legal limits for them, will affect the scope and range of the risk assessment process. The chemicals of concern must be identified prior to any data collection.

6.6 RISK COMMUNICATION

6.6.1 PUBLIC PERCEPTION OF RISK

Risk perception is a lot more than mortality statistics. If death rates are the only thing you care about, then the public is afraid of the wrong risks. That is, public fears are not well correlated with expert assessments or mortality statistics. This is often seen as a perceptual distortion on the part of the public, but a more useful way to see it is as an oversimplification on the part of many experts and policy-makers. In other words, the concept of "risk" means a lot more than mortality statistics.

Virtually everyone would rather drive home from a party on the highway than walk home on desert streets. Even if we do not miscalculate the relative statistical likelihood of a fatal mugging versus a fatal car crash, the possibility of getting mugged strikes us as an outrage, while we accept the possibility of an auto accident as voluntary and largely controllable through good driving. (Eighty-five percent of all drivers consider themselves better than average.) Similarly, a household product, however carcinogenic, seems a lot less risky than a high-tech hazardous waste treatment facilityCthe former is familiar and under one's own control, while the latter is exotic and controlled by others.

Risk perception experts have spent years studying how people interpret

risk. The following list identifies some of the characteristics other than mortality that factor into our working definitions of risk. Remember, these are not distortions of risk; they are part of what we mean by the term.

Less Risky	More Risky
Voluntary	Involuntary
Familiar	Unfamiliar
Controllable	Uncontrollable
Controlled by self	Controlled by others
Fair	Unfair
Not memorable	Memorable
Not dread	Dread
Chronic	Acute
Diffuse in time and space	Focused in time and space
Not fatal	Fatal
Immediate	Delayed
Natural	Artificial
Individual mitigation possible	Individual mitigation impossible
Detectable	Undetectable

It doesn't help to wish to people would confine their definitions of risk to the mortality statistics. They won't. Mortality statistics are important, of course, and policy-makers understandably prefer to focus on the risks that are really killing people, rather than the risks that are frightening or angering people because they are involuntary, unfamiliar, uncontrollable, etc. But successful risk communication begins with the realization that risk perception is predictable, that the public overreacts to certain sorts of risks and ignores others, and that you can know in advance whether the communication problem will be related to panic or apathy. And since these differences between risks are real and relevant, it helps to put them on the table. Merely acknowledging that a risk seems especially fearful because it is unfamiliar or unfair will help. Doing something to remedy the unfamiliarity or unfairness will help even more.

Risk judgments are also very responsive to verbal cues. Doctors, for example, are much more likely to prescribe a new medication that saves 30 percent of its patients than one that loses 70 percent of them. A pollutant or an accident that will eventually give cancer to 10,000 people sounds very serious, but one that will add less

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than one tenth of one percent to the national cancer rate sounds almost negligible. There is in fact no "neutral" way to present risk data, only ways that are alarming or reassuring in varying degrees.

6.6.2 MORALITY VS. RISK

Moral categories mean more than risk data. The public is far from sure that risk is the real issue in the first place. Over the past several decades our society has reached near-consensus that pollution is morally wrong—not just harmful or dangerous, not just worth preventing where practical, but wrong. To many ears it now sounds callous, if not immoral, to assert that cleaning up a river or catching a midnight dumper isn't worth the expense, that the cost outweighs the risk, that there are cheaper ways to save lives. The police do not always catch child molesters, but they know not to argue that an occasional molested child is an "acceptable risk." An agency that wishes to deal with environmental risk in terms of costs-and-benefits instead of good-and-evil should proceed gently and cautiously, aware that it is tromping on holy ground. Just as the moralist challenges the rightness of trading off certain risks against costs or benefits, the humanist challenges the coherence of the tradeoffs. How, the humanist asks, can anyone make sense of a standard that tries to put a cash value on human life? Or, indeed, of a standard that assumes that a hundred widely scattered deaths per year are equivalent to a one-in-a-hundred chance of obliterating a community of 10,000?

Similarly, the political critique of the premises of risk assessment begins by noting that "the greatest good for the greatest number" has always been a convenient rationale for the oppression of minorities. Democratic theory asserts that individuals and groups should be free to bargain for their own interests, and should be protected from the tyranny of the majority. There is nothing unreasonable about the suggestion that equitable distribution of risks and benefits—and of the power to allocate risks and benefits—is often more important than the minimization of total risk or the maximization of total benefit. It may be efficient to dump every environmental indignity on the same already degraded community, but it is not fair.

6.6.3 RISKY OR SAFE?

Policy decisions are seen as either risky or safe. Like the media, the public tends to dichotomize risk. Either the risk is seen as very frightening, in which case the response is some mix of fear, anger, panic, and paralysis; or the risk is dismissed as trivial, in which case the response is apathy. While people may (with difficulty) master a

probabilistic risk statement that concerns what they should do to protect themselves, they are bound to resist probabilistic risk statements that concern what others (government, say) should do to protect them.

Quantitative risk assessments, risk-benefit calculations, risk-cost ratios, and risk-risk comparisons are all hard to hear when we bear the risk and someone else make the decision.

6.6.4 CONTROL OF RISK

Equity and control issues underlie most risk controversies. Trust and credibility are often cited as the key problems of risk communication. Certainly few people trust government and industry to protect them from environmental risk. This is just as true of the passive, apparently apathetic public as it is of the activist, visibly angry public. The former is simply more fatalistic, more prone to denial, more completely drowned in indiscriminating chemophobia.

The activist public, in other words, distrusts others to protect its interests and thus chooses to protect its own. The far larger passive public is passive not because it believes others will protect its interests, but because it doubts it can protect its own. Both publics listen to the reassurances of government and industry if they listen at all with considerable suspicion.

But to say that trust is the problem here is to assume that the goal is a passive public that doesn't mind being passive. If the goal is an actively concerned public, then the problem isn't that people are distrustful, but rather that government and industry demand to be trusted. Translate the question of trust into the underlying issue of control: Who decides what is to be done?

Any environmental risk controversy has two levels. The substantive issue is what to do; the process issue is who decides. So long as people feel disempowered on the process issue, they are understandably unbending on the substantive issue, in much the same way as a child forced to go to bed protests the injustice of bedtime coercion without considering whether he or she is sleepy. It isn't just that people oppose any decision they view as involuntary and unfair, regardless of its wisdom; because the equity and control issues come first, people typically never even ask themselves whether they agree on the merits. Outraged at the coercion, they

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simply dig in their heels. It is hardly coincidental that risks the public tends to overestimate generally raise serious issues of equity and control, while most of the widely underestimated risks (smoking, fat in the diet, insufficient exercise, driving without a seatbelt) are individual choices.

The gravest problems of risk communication tend to arise when citizens determine that the issue is important, that the authorities cannot be trusted and that they themselves are powerless. Then comes the backlash of outrage.

6.6.5 SHARE THE POWER

Risk decisions are better when the public shares the power. People learn more and assess what they learn more carefully if they exercise some real control over the ultimate decision. But this sort of power-sharing is, of course, enormously difficult for policy-makers, for a wide range of political, legal, professional, and psychological reasons. Interestingly, corporate officials may sometimes find power-sharing less unpalatable than government officials. Corporations have a bottom line to nurture, and when all else fails they may see the wisdom of sharing power in the interests of profit. But government officials have no profit to compensate for the loss of power, so they may find it harder to share. Most public participation is too little too late: "After years of effort, summarized in this 300-page report, we have reached the following conclusions...Now what do you folks think?" At this point it is hard enough for the agency to take the input seriously, and harder still for the public to believe it will be taken seriously. There is little power-sharing in the "decide-announce-defend" tradition of public participation.

The solution is obvious, though difficult to implement. Consultations with the public on risk management should begin early in the process and continue throughout. When citizens participate in a risk management decision, moreover, they are far more likely to accept it, for a least three reasons: (1) They have instituted changes that make it objectively more acceptable; (2) They have got past the process issue of control and mastered the technical data on risk; that is they have learned why the experts consider it acceptable; and (3) They have been heard and not excluded, and so can appreciate the legitimacy of the decision even if they continue to dislike the decision itself.

In many risk communication interactions, in short, the public doesn't really want to understand (because it feels powerless and resentful) and the experts don't really want to be understood

(because they prefer to hold onto their information monopoly). The public finds it convenient to blame the experts for obfuscation, and the experts find it convenient to blame the public for obtuseness. These motivational issues are probably more important than the traditional concerns of clarity in determining whether real knowledge will pass from expert to public.

Within the traditional concerns of clarity, the major issue is simplification. Even assuming a public that wants to understand and an expert who wants to be understood, risk information must still be simplified. Insofar as possible, of course, it is wise to simplify language rather than content. That is, take the extra words to make hard ideas clear.

6.6.6 COMMUNICATION

In fact, there are three standard rules of thumb for popularizing technical content. (1) Tell people what you have determined they ought to know—the answers to the questions they are asking, the instructions for coping with the crisis, whatever. (2) Add what people must know in order to understand and feel that they understand the information—whatever context or background is needed to prevent confusion or misunderstanding. (3) Add enough qualifiers and structural guidelines to prepare people for what you are not telling them, so additional information later will not leave them feeling unprepared or misled.

The hardest part of simplifying risk information is explaining the risk itself. This is hard not only because risk assessments are intrinsically complex and uncertain, but also because audiences cling tenaciously to their safe-or dangerous dichotomy. One path out of dichotomous thinking is the tradeoff: especially risk benefit, but also risk-cost or risk-risk. But there is solid evidence that lay people resist this way of thinking; trading risks against benefits is especially offensive when the risks raise moral issues and the "victims" are not the ones making the choice. Another alternative to dichotomy is the risk comparison: X is more dangerous than Y and less dangerous than Z. But as we have already noted, risk means a lot more than mortality statistics, and comparing an involuntary risk like nuclear power to a voluntary one like smoking invariably irritates more than it enlightens—as does any risk comparison that ignores the distinctions listed at the start of this section.

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The final option to dichotomy is to provide the actual data on deaths or illnesses or probability of occurrence or whatever. This must be done carefully, with explicit acknowledgement of uncertainty, of moral issues, and of non-statistical factors like voluntariness that profoundly affect our sense of risk. Graphs and charts will help; people understand pictorial representations of probability far better than quantitative ones. Over the long haul, risk communication has more to do with fear, anger, powerlessness, optimism and overconfidence than with finding ways to simplify complex information. Many have spent years learning to ignore feelings, their own and everyone else's; whether they are scientists interpreting data or managers setting policy, they are deeply committed to doing their jobs without emotion.

Thus the most common sources of risk information are people who are professionally inclined to ignore feelings. And how do people respond when their feelings are ignored. They escalateC yell louder, cry harder, listen lessC which in turn stiffens the experts, which further provokes the audience. The inevitable result is the classic drama of stereotypes in conflict: the cold scientist or bureaucrat versus the hysterical citizen.

Breaking this self-defeating cycle is mostly a matter of explicitly acknowledging the feeling (and the legitimacy of the feeling) before trying to explain anything substantiveC because any effort to explain substance first will be experienced by people as just another way of not noticing how they feel. The trick, in other words, is to separate the feeling from the substance, and respond to the feeling first.

Feelings are not usually the core issue in risk communication controversies. The core issue is usually control, and the way control affects how people define risk and how they approach information about risk. But the stereotypical conflict between the icy expert and the hysterical citizen is nonetheless emblematic of the overall problem. The expert has most of the "rational" resourcesC expertise, of course; stature; formal control of the ultimate decision. Neither a direct beneficiary nor a potential victim, the expert can afford to assess the situation coldly. Indeed, the expert dare not assess the situation in any other way. The concerned citizen, meanwhile, has mainly the resources of passionC genuine outrage; depth of commitment; willingness to endure personal sacrifice; community solidarity; informal political power. To generate the energy needed to stop the technical juggernaut, the citizen must assess the situation hotly.

A fundamental premise of "Explaining Environmental Risk" is that

risk understanding and risk decision-making will improve when control is democratized. We will know this happening when citizens begin approaching risk issues more coolly, and experts more warmly.

6.7 ADDITIONAL READING

EPA: "Risk Assessment Guidance for Superfund, Vol. 1, Human Health Evaluation Manual (Part A)", EPA/540/1-89/002, Dec. 1989 and "Risk Assessment Guidance for Superfund, Vol. 2, Environmental Evaluation Manual, Interim Final, EPA/540/1-89/001, March 1989).

6.8 GLOSSARY

Absorption: The uptake of water or dissolved chemicals by a cell or an organism.

Absorption Factor: The fraction of a chemical making contact with an organism that is absorbed by the organism.

Acceptable Daily Intake (ADI): A term formerly used to refer to an estimated exposure level that would not result in adverse health effects.

Acceptable Intake Chronic (AIC): The highest human intake of a chemical, expressed as mg/kg/day, that is not expected to cause adverse effects when exposure is long-term (lifetime).

Acceptable Intake Subchronic (AIS): The highest human intake of a chemical, expressed as mg/kg/day, that is not expected to cause adverse effects when exposure is short term (but not acute).

Acute: Occurring over a short period of time; used to describe brief exposures and effects which appear promptly after exposure.

Agency for Toxic Substances and Disease Registry (ATSDR): Agency which has primary responsibility for health assessments at Superfund sites.

Ambient Water Quality Criteria (AWQC): The health based water quality criterion is an estimate of the ambient surface water concentration that shall not result in adverse effects in humans.

Applicable or Relevant and Appropriate Requirements (ARARs):

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Applicable requirements are cleanup standards, standards of control, and other environmental protection requirements promulgated by law that address contaminants at Superfund sites. Relevant and appropriate requirements are cleanup standards, standards of control, and other protection requirements while not applicable to hazardous substance, contaminant, remedial action, location, or other circumstances at CERCLA sites, address problems or situations sufficiently similar to those encountered at the Superfund site such that their use is well suited to the particular site.

Cancer: A general term frequently used to indicate any of various type of malignant neoplasms, most of which invade surrounding tissues, may metastasize to several sites, and are likely to recur after attempted removal and to cause death of the patient unless adequately treated.

Cancer Potency Factor: The upper 95% confidence limit (when based on animal data) or the maximum likelihood estimate (when based on human data) on the slope of the dose-response curve expressed in units of $(\text{mg/kg/day})^{-1}$.

Carcinogen: An agent or substance capable of inducing a tumor. Includes both those substances that produce benign tumors as well as malignant tumors.

Chronic: Occurring over a long period of time; used to describe ongoing exposures and effects that usually develop only after long exposure periods.

Contaminants of Concern: Refers to the subset of contaminants identified at a Superfund site, that are selected for risk characterization based on their toxicity, concentration, frequency of detection, persistence and mobility.

Dose: The quantity of a chemical to which an organism is exposed.

Drinking Water Equivalent Level (DWEL): Established by EPA's Office of Drinking Water to protect against adverse health effects resulting from a lifetime of exposure to noncarcinogenic contaminants in drinking water.

Environmental Transport Medium: Mode of moving contaminant (air, ground water, etc).

Epidemiological Studies: Investigation of elements contributing to disease or toxic effects in human populations.

Exposure: Contact with a chemical or physical agent.

Exposure Pathway: An exposure pathway consists of four elements: (1) a source and mechanism of chemical release to the environment, (2) an environment transport medium, (3) a point of potential human contact with the contaminated medium, (4) a human exposure route at the contact point.

Hazard Index (HI): The term used to describe the potential for noncarcinogenic health effects. It is computed by dividing the exposure dose by the reference dose or other suitable noncarcinogenic standard or criteria for an individual chemical.

Health Effects Assessments: A group of reports prepared by EPA which present a brief summary and evaluation of information relevant to a preliminary interim assessment of the adverse health effects of various chemicals.

Hematopoietic System: The system responsible for producing and maintaining the constituents of blood. Includes the circulating blood, lymphoid tissue, and the bone marrow.

Hepatic: Associated with supplying or draining the liver.

Histopathology: A branch of pathology concerned with the tissue changes characteristic of disease.

Indicator Compounds: See contaminants of concern.

Integrated Risk Information System (IRIS): Computer database which provides updated dose-response information for many chemicals. Represents the principal resource for current EPA dose-response information.

International Agency for Research on Cancer (IARC): Non-regulatory agency involved in cancer research and which developed an approach to classifying the cancer weight evidence based on human and animal data.

Lifetime Health Advisory (HA): Guidelines established by the EPA Office of Drinking Water to protect against noncarcinogenic health effects of compounds in drinking water. Derived strictly based on health considerations.

Lowest Observable Adverse Effect Level (LOAEL): The lowest

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dose in an experiment which produced an observable adverse effect.

Maximum Contaminant Levels (MCL): Enforceable chemical specific standards set under the Safe Drinking Water Act for public water supplies. MCLs are based on health, treatability, and cost considerations.

Maximum Contaminant Level Goal (MCLG): Non-enforceable chemical specific drinking water concentrations established under the Safe Drinking Water Act that are entirely health based.

Metabolism: The sum of the chemical reactions occurring within a cell or a whole organism; includes the energy-releasing breakdown of molecules (catabolism) and the synthesis of new molecules (anabolism).

Metabolite: Any product of metabolism, especially a transformed chemical.

Mutagenicity: The capacity of a chemical or physical agent to cause permanent alteration of the genetic material within living cells.

No Observable Adverse Effect Level (NOAEL): The highest dose in an experiment which did not produce an observable adverse effect.

Non-detect (ND): The term used to refer to a chemical that is not present in sufficient quantity to be accurately quantified.

Pica: An abnormal desire to eat non-food substances, especially in children up to age 6. .

Receptor: An organism that receives, may receive, or has received environmental exposure to a chemical.

Reference Dose (RfD): The RfD is based on EPA's identification of the threshold effects level with an added margin of safety. The RfD represents an estimate of the daily dose level (mg/kg/day) for a particular compound that is likely to be without appreciable risk of deleterious effect when exposure occurs over a given period (usually a lifetime).

Relative Absorption Factor: The ratio of the estimated absorption factor for the site specific medium and route of exposure to the known or estimated adsorption factor for the laboratory study from which the cancer potency factor or reference dose was derived.

Risk: The potential for realization of unwanted negative consequences or events.

Suggested No Adverse Response Level (SNARL): A term formerly used by EPA and the National Academy of Sciences corresponding to a contaminant level in drinking water at which adverse health effects would not be anticipated.

Superfund Public Health Evaluation Database (PHRED): Computer database available through EPA with chemical, physical, and toxicity information on various compounds.

Teratogenicity: The capacity of a physical or chemical agent to cause non-hereditary congenital malformations (birth defects) in offspring.

Toxic/Target-Endpoint: The most sensitive yet significant noncarcinogenic effect caused by the administration of a compound. Examples include enzyme, weight, gross morphological, and functional changes. Also the target endpoint serves as the basis from which some of the noncarcinogenic criteria (RfDs) are derived.

Toxicity: The quality or degree of being poisonous or harmful to plant, animal or human life.

Uncertainty Factor (U.F.): A factor used to account for the interspecies and intraspecies differences, severity of adverse effects, and the adequacy of data when determining the reference dose.

Xenobiotic: A chemical compound that is foreign to a living organism.

TABLE 4{PRIVATE }

Recommended Approaches for Addressing Key Questions for Ecological Assessments at Hazardous Waste Sites

Key Questions	Recommended Approach	Example Measurement Endpoints and Outputs
Have biological communities or populations, on site or off site, been measurably impacted at the HWS?	Field surveys	Occurrence and abundance of important species at the HWS relative to values for comparable reference areas.
Are soils, water, or sediments at the HWS contaminated?	Chemical analysis	Chemical concentrations of contaminants of concern, at the HWS, relative to values for comparable reference areas.
		Toxic response to samples.
Are the contaminated soils, water, and sediments at the HWS toxic or hazardous to living organisms?	Toxicity tests	Percent survival or occurrence of biomarkers for organisms exposed to contaminated media for the HWS, relative to appropriate reference values.
	Acute and chronic toxicity tests	
	Biomarkers of sublethal stress	Chemical concentrations of contaminants or frequency of occurrence of other biomarkers or organisms collected from the field at the HWS, relative to values for organisms from comparable reference areas.
Are organisms at the HWS exposed to these hazardous contaminants?	Biomarkers of exposure	
		Comparison of the spatial patterns for effects at the HWS measured with (1) field surveys of ecological status, toxicity testing with contaminated media, (2) surveys of bio-markers of exposure and sub-lethal stress, (3) chemical surveys, and (4) outputs from fate and transport modeling.
Are the effects of biological communities and the populations at the HWS caused by the presence of hazardous wastes?	Use of all of the above	